Pancreatitis in systemic lupus erythematosus – a case report and review of the literature

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Summary

A young woman with a past history of haemolytic anaemia was admitted to the hospital for pancreatitis and found to have serological evidence of systemic lupus erythematosus (SLE). The occurrence of pancreatitis as a complication of SLE is reviewed.

Introduction

Gastrointestinal manifestations including abdominal pain, diarrhoea, nausea and vomiting have been described in patients with systemic lupus erythematosus (SLE). Of the 520 patients surveyed by Dubois and Tuffanelli (1964), 53% displayed gastrointestinal symptoms. These symptoms are caused most probably by disseminated vasculitis (Brown, Shirey and Haserick, 1956; Dubois, 1953; Pollak *et al.*, 1958; Weiser and Harist, 1978).

Acute pancreatitis in SLE has been reported in a small number of cases, (Dubois, 1953; Paulino-Netto and Dreiling, 1960; Pollak *et al.*, 1958; Sparberg, 1967). Some of the patients described in those publications were treated with steroids and/or diuretics which could have been an added factor in the development of pancreatitis (Johnston and Cornish, 1959; Oppenheimer and Boitnott, 1960; Penso *et al.*, 1973). In this report a case is presented in which acute pancreatitis appeared together with the first serological evidence of SLE. Autoimmune haemolytic anaemia had preceded these manifestations by 13 years.

Case report

A 32-year-old female of Arab origin was referred to the Meir Hospital, Tel Aviv, following an attack of severe abdominal pain radiating to the back, accompanied by vomiting with bile content. This patient was known to suffer from recurrent auto-

immune haemolytic anaemia with positive direct and indirect Coombs' tests without an apparent aetiology. The haemolytic attacks responded well to steroid treatment. L.E. cells and antinuclear antibodies (ANA) were negative during this period. Following 5 years of maintenance steroid therapy the haemoglobin count remained continuously around 13 g/dl and treatment was discontinued. For several years the patient had no clinical complaints but during the past year intermittent abdominal pain appeared, following one of which she was admitted to the medical ward. No medication was taken during this period.

Examination revealed an obese woman who seemed acutely ill and in pain. No jaundice was apparent. The temperature was 37°C, the pulse 82, BP was 130/80 mmHg. No rash or lymphadenopathy was found; the optic fundi were normal. The lungs and heart were normal, no pleural or pericardial friction rub was heard. Abdominal examination disclosed a soft abdomen with diffuse mild tenderness and considerable voluntary guarding; no organ, mass or spasm was felt.

Laboratory findings: Hb – 13.9 g/dl; WBC – $5.0 \times 10^9/l$ with a normal differential count; platelet count of $15 \times 10^{10}/l$; ESR – 56 mm in the 1st hr (Westergren). Serum glucose level was 6.0 mmol/l; urea – 4 mmol/l and creatinine – 88.4 $\mu\text{mol/l}$. Creatinine clearance was 70 ml/min. Transaminases, alkaline phosphatase, cholesterol, triglycerides and serum electrolytes were all within the normal limits. Urine amylase was 5500 i.u. while in the blood it was 900 i.u. The amylase to creatinine clearance ratio (Cam/Ccr) was 7.4. Proteinuria (4 g/day) was noticed. L.E. cells were found and ANA were positive at a titre of 1 : 5000. C_3 level was 53 mg/dl, no antibodies against double-stranded DNA were found. A stool specimen gave a negative test for occult blood.

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The diagnosis of acute pancreatitis was made and treatment was given accordingly. Owing to the possibility that the pancreatitis was a complication of SLE, 60 mg of prednisone/day were given, resulting in the disappearance of all the symptoms. Amylase levels returned to normal values. An X-ray of the upper gastrointestinal tract (GIT) and a barium enema revealed no pathology except for a slight distension of the small intestine. Cholangiography showed normal bile ducts and gall bladder.

To rule out diffuse vasculitis a jejunal biopsy was performed using a Crosby capsule. The histological findings disclosed a normal mucosa and submucosa. No immunofluorescent deposits of immunoglobulin and C₃ were detected. A kidney biopsy revealed local focal glomerulitis.

The steroid dosage was gradually decreased to 20 mg prednisone/day, the patient was discharged free of symptoms.

Discussion

In SLE symptoms of gastrointestinal involvement appear with relatively high frequency. Abdominal pain occurs in about 20% of the patients while nausea and vomiting occur in more than 50% (Dubois and Tuffanelli, 1964). Although there are many reports of GIT involvement in SLE, histological data are still limited to post-mortem examinations. This is due in most cases to the fact that the symptoms are relatively mild and do not require surgical intervention. The common histological finding in these reports is disseminated vasculitis in various gastrointestinal organs which causes infarcts, ulcers, perforations and peritonitis (Jarcho, 1936; Harvey *et al.*, 1954; Pollak *et al.*, 1958; Weiser and Harrist, 1978; Zizik, Shulman and Steves, 1975).

Despite all the above examples acute pancreatitis is a rare complication of SLE. The clinical picture of the patient was accompanied by a number of findings characteristic of acute pancreatitis: (a) increased levels of amylase were evident in the blood and urine. Amylase to creatinine clearance ratio was 7.4, values >4 are characteristic of pancreatitis (Salt and Schenker, 1976); (b) there was a slight distension of intestine loops in abdominal X-ray.

There was no evidence of gall stones and the possibility of hyperlipidaemia as a cause of pancreatitis was ruled out. The patient did not take any medication which could have caused pancreatitis during the last 3 months before her admission. An important observation in this case was the impressive response of the patient to steroid treatment which supported the concept of a SLE aetiology.

The common findings in SLE-induced pancreatitis are related to inflammatory responses in pancreatic arteries which are similar to vasculitis in other areas

of the GIT. Pollak and his colleagues (1958) have described 4 cases of SLE-associated pancreatitis of which 3 patients died and were examined *post mortem*. In one case, the lesions in the large vessels of the pancreas resembled those found in periarteritis nodosa. In the second case small arteries and arterioles were occluded by thrombi, which resembled thrombotic thrombocytopenic purpura (TTP). In the third case, acute pancreatitis was noted in association with end-stage renal failure. Dubois (1953) reported a SLE patient with severe epigastric pain radiating to the back, displaying a serum amylase concentration level of 2400 i.u. Histopathological examination of the pancreas revealed local arteritis. A similar finding of vasculitis and chronic sclerosis with parenchymal atrophy was reported by Seifert, Heinz and Ruffman (1967) in a SLE patient with pancreatitis which was not treated with steroids.

It is conceivable that the pancreatitis noted in SLE is a result of immune complex deposits and complement activation in the walls of pancreatic arteries (Seelig and Seelig, 1975).

Of interest is the atypical clinical course of this patient – a long-standing history of autoimmune haemolytic anaemia without serological evidence of SLE. With the disappearance of the haemolytic anaemia, gastrointestinal symptoms together with serological evidence of SLE were found. One can only speculate that because of the prolonged steroid therapy the serological manifestation of SLE was masked (Dubois, 1974).

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